In the Specification:

Please replace the paragraph on page 4, lines 11-20, with the following rewritten paragraph:

The present invention is directed to a method and pharmaceuticals for treating HIV and secondary infection. One aspect of this invention involves the use of one or more polypeptides with an amino acid sequence including KPV, MEHFRWG (SEQ ID NO: 1), HFRWGKPV (SEQ ID NO: 2), or SYSMEHFRWGKPV (SEQ ID NO: 3) for treatment of HIV. HIV is accompanied by infections, inflammation or both. In one preferred embodiment of the invention, the one or more polypeptides are used for treatment of HIV itself via medication taken orally or parenterally. In another preferred embodiment of the invention, the treatment is for secondary infections arising from *Staphylococcus aureus* and *Candidia albicans* and can be taken either orally or parentally. In another preferred embodiment of the invention, treatment is carried out by local application of the polypeptides through a carrier onto the site of *S. aureus* or *C. albicans* infection.

Please replace the paragraph on page 7, lines 8-9, with the following rewritten paragraph:

Fig. 16 shows a representation of the chemical structure for the amino acid sequence VPKCCKPV (SEQ ID NO: 4), known as the "KPV dimer."

Please replace the paragraph on page 7, line 21, to page 8, line 13, with the following rewritten paragraph:

α-MSH (SYSMEHFRWGKPV) (SEQ ID NO: 3) is a pro-opiomelanocortin (POMC)-derived tridecapeptide with potent antipyretic and anti-inflammatory influences. The C-terminal tripeptide of α-MSH, KPV exerts anti-inflammatory effects in vitro and in

animal models of inflammation, that are similar to those of the entire 1-13 sequence. Therefore, KPV is considered the anti-inflammatory "message sequence" of the peptide. Five G-protein-linked melanocortin receptors (MC-IR through MC-5R) have been isolated and cloned. They bind α-MSH and other melanocortin peptides such as ACTH with different affinities; MC-R are expressed in the brain and in peripheral tissues. The precursor of α-MSH, POMC, is widely expressed in tissues. However, there are distinct regional differences in expression and post-translational processing and certain cells produce, constitutively or under appropriate stimulation, greater amounts of α-MSH. α-MSH occurs in high concentrations in barrier organs such as the gut and the skin. Activated macrophages and microglia likewise produce substantial amounts of α-MSH and there is evidence that the peptide has an autocrine anti-inflammatory influence in these cells that express melanocortin receptors. The anti-inflammatory effects of α-MSH are exerted partly via inhibition of certain inflammatory mediators, such as cytokines and nitric oxide, likely through inhibition of the transcription factor NF-kB.

Please replace the paragraph on page 9, lines 4-5, with the following rewritten paragraph:

α-MSH 1-13 SYSMEHFRWGKPV (SEQ ID NO: 3) and (11- 13) KPV, both acetylated and amidated, were used.

Please replace the paragraph on page 11, line 8, to page 12, line 8, with the following rewritten paragraph:

For determination of NF-kB activity, nuclear extracts were prepared from 20x 10^6 UI cells (2 x 10^5/mL in complete medium) stimulated for 4 h with TNF-α^{32} (20 ng/mL) in the presence or absence of 10^{-5} M KPV. Cells were washed once with cold PBS, and

twice with buffer A (10 mM Hepes pH 7.9, 1.5 mM $MgCl_2$, 10 mM KCl, 0.5 mM phenylmethylsulfonyl fluoride [PMSF; Boehringer Mannheim] and 0.5 mM dithiothreitol (DTT) [Sigma-Aldrich]), centrifuged and incubated for 10 min on ice in buffer A plus 0.1% NP-40 (Sigma-Aldrich). The supernatants were removed and the nuclear pellets resuspended in 15 μl of buffer C (20 mM Hepes pH 7.9, 1.5 mM $MgC1_2$, 0.42 M KCl, 0.2 mM EDTA, 25% glycerol, 0.5 mM PMSF and 0.5 mM DTT), incubated for 15 min on ice, mixed and centrifuged. The supernatants were diluted with 75 μl of modified buffer D (20 mM Hepes pH 7.9, 0.05 mM KC1, 0.2 mM EDTA, 20% glycerol, 0.5 mM PMSF and 0.5 mM DTT) and stored at -80°C. The binding reaction was carried out with 10 μg of nuclear extract protein and 0.5 ng of α^{32} P-labeled NF-kB (30,000 cpm/μl) or AP1 consensus in buffer A (12 mM Tris-HCl pH 7.8, 60 mM KCl, 0.2 mM EDTA, 0.3 mM DTT), plus 10% glycerol, 2μg/mL bovine serum albumine (BSA, Sigma-Aldrich) and lμg/mL sDNA (Pharmacia Biotech), for 15 min at room temperature. The oligonucleotides for NF-kB used in these studies were: + GAT CCA AGG GGA CTT TCC GCT GGG GAC TTT CCA TG, (SEQ ID NO: 5) and - GAT CCA TGG AAA GTC CCC AGC GGA AAG TCC CCT TG (SEQ ID NO: 6). Each oligonucleotide was annealed to its complementary strand and end-labeled with α^{32}P-γ-ATP (Amersham Corp., Arlington Heights, IL) using polynucleotide kinase (T4, New England Biolabs). For determination of specific bands, nuclear extracts were first incubated with 100-fold excess unlabeled probe for 5 min before incubation with the labeled probe. The mixtures were run on a 5% (30:1) acrylamide gel in 1x Tris-Borate-EDTA (TBE). Gels were dried and exposed to film for autoradiography (3 days).

Please replace the paragraph on page 12, line 10, to page 13, line 5, with the following rewritten paragraph:

U1 cells were seeded in T25 flasks at a density of $2x10^5$ cells/mL and incubated with medium alone or PMA (1 ng/mL) for 24 h. Cells were then washed with cold PBS and total RNA was extracted as described above. The amount of total RNA was determined photometrically at 260 nm. Extracted RNA was used for reverse transcription and PCR amplification. Because MC-1 receptor gene lacks introns, genomic DNA was digested with DNAse for 30 min at 37°C. DNAse was inactivated by phenol-chloroform extraction. One microgram of total RNA was reverse transcribed using random primers and AMV RT (Boehringer Mannheim). In some tubes the RT was omitted to control for amplification from contaminating cDNA or genomic DNA. Fragments derived from the coding regions of human MC-1 cDNAs were amplified using primers synthesized commercially (Genset SA, France). The MC-IR forward primer was GCC ACC ATG CCA AGA ACC, (SEQ ID NO: 7) the reverse primer was ATA GCC AGG AAG AAG ACCA (SEQ ID NO: 8) (all shown as 5' to 3'). PCR mixture contained the following: 0.8 μM of each primer, 1.5 mM $MgCl_2$, 200 μM dNTPs, 10 x reaction buffer and 2 units of Taq DNA polymerase/reaction (AmplyTaq; Perkin Elmer Italiana, Monza, Italy). To minimize nonspecific amplification, the Taq DNA polymerase was added to PCR tubes prewarmed to 80°C. The PCR temperature profile consisted of 35 cycles of 94°C for 45 sec (denaturing), 57°C for 45 sec (annealing), and 72°C for 1 min, followed by a 7-min final extension at 72°C. The PCR products were separated on 2% agarose, stained with ethidium bromide and photographed under UV light.

Please replace the paragraph on page 20, lines 4-10, with the following rewritten paragraph:

The peptides used in this research included: α-MSH (1-13), (4-10), (6-13) and (11-13), all of which were N-acetylated and C-amidated and ACTH (1-39) and (18-39) (CLIP). Another peptide used in this research included a dimer of the amino acid sequence KPV, specifically VPKCCKPV (SEQ ID NO: 4), which also was N-acetylated and C-amidated (the "KPV dimer"). The KPV dimer can be chemically represented as NH_2-Val-Pro-Lys-Ac-Cys-Cys-Ac-Lys-Pro-Val-NH_2~~NH_3~~ (SEQ ID NO: 4). The peptides were prepared by solid-phase peptide synthesis and purified by reversed-phase high performance liquid chromatography.

Please replace the paragraph on page 23, line 21, to page 24, line 3, with the following rewritten paragraph:

α-MSH peptides (1-13) and (11-13) inhibited *S. aureus* colony formation (Fig. 1). A dimer of the amino acid sequence KPV, specifically, ~~NH_2-Lys-Pro-Val-Ac-Cys-Cys-Ac-Val-Pro-Lys-NH_2~~ NH_2-Val-Pro-Lys-Ac-Cys-Cys-Ac-Lys-Pro-Val-NH_2 (the "KPV dimer") (SEQ ID NO: 4)also inhibited *S. aureus* colony formation (Fig. 8). The inhibitory effect occurred over a wide range of concentrations and was significant ($p < 0.01$) with peptide concentrations of 10^{-12} to 10^{-4}M.

Please replace the paragraph on page 24, lines 8-11, with the following rewritten paragraph:

C. albicans colony forming units ("CFU") were greatly reduced by α-MSH (1-13) and (11-13) (Fig. 10). A dimer of the amino acid sequence KPV, specifically, ~~KPVCCVPK~~ NH_2-Val-Pro-Lys-Ac-Cys-Cys-Ac-Lys-Pro-Val-NH_2 (the "KPV dimer") (SEQ

ID NO: 4) also inhibited *C. albicans* colony formation (Fig. 10). Concentrations of all three peptides from 10^{-13} to 10^{-4}M had significant inhibitory influences on CFU ($p<0.01$ vs. control).

Please replace the paragraph on page 26, lines 3-14, with the following rewritten paragraph:

The results show that α-MSH (1-13), its C-terminal tripeptide (11-13), and other α-MSH fragments have significant antimicrobial effects against at least two major pathogens: *S. aureus* and *C. albicans*. The most effective of the α-MSH peptides were those including the C-terminal amino acid sequence KPV of the α-MSH sequence, i.e., α-MSH (1-13), (6-13) and (11-13). A dimer of the amino acid sequence KPV, specifically, VPKCCKPV (SEQ ID NO: 4) (referred to herein as the "KPV dimer," shown in Fig. 16) has also been shown to be at least as effective as α-MSH (11-13) against microbes. The α-MSH "core" sequence (4-10), which is known to influence learning and memory, but has little antipyretic and anti-inflammatory influence, was effective, but less so. The ACTH peptides (1-39) and (18-39) did not have significant candidacidal effects. These observations indicate that antimicrobial activity is not common to all melanocortin peptides, but rather that it is specific to α-MSH amino acid sequences, and most particularly to the C-terminal amino-acid sequences of α-MSH.

Please replace the paragraph on page 32, line 18, to page 33, line 4, with the following rewritten paragraph:

Polypeptides that include an amino acid sequence from the group consisting of KPV, MEHFRWG (SEQ ID NO: 1), HFRWGKPV (SEQ ID NO: 2), and SYSMEHFRWGKPV (SEQ ID NO: 3) are useful in pharmaceutically acceptable oral,

parenteral or topical modes. These pharmaceutical compositions contain an amino acid sequence from the group consisting of KPV, MEHFRWG (SEQ ID NO: 1), HFRWGKPV (SEQ ID NO: 2), and SYSMEHFRWGKPV (SEQ ID NO: 3) in association with a compatible pharmaceutically acceptable carrier material. Any conventional carrier material can be utilized. The carrier can be organic or an inert inorganic carrier. For oral use, the carrier may be water, gelatin, gum arabic, lactose, starch, magnesium stearate, talc, vegetable oils, polyalkylene-glycols, petroleum jelly and the like. Additional additives, such as flavorings, preservatives, stabilizers, emulsifiers, buffers and the like may be added in accordance with accepted practices of pharmaceutical compounding.